



## Dissociative symptoms and sleep parameters — an all-night polysomnography study in patients with insomnia

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### Abstract

**Background:** Dissociative disorders encompass a range of symptoms varying from severe absent-mindedness and memory problems to confusion about one's own identity. Recent studies suggest that these symptoms may be the by-products of a labile sleep–wake cycle.

**Methods:** In the current study, we explored this issue in patients suffering from insomnia ( $N=46$ ). We investigated whether these patients have raised levels of dissociative symptoms and whether these are related to objective sleep parameters. Patients stayed for at least one night in a specialized sleep clinic, while sleep EEG data were obtained. In addition, they completed self-report measures on dissociative symptoms, psychological problems, and sleep characteristics.

**Results:** Dissociative symptom levels were elevated in patients suffering from insomnia, and were correlated with unusual sleep experiences and poor sleep quality. Longer REM sleep periods and less time spent awake during the night were predictive of dissociation.

**Conclusions:** This is the first study to show that insomnia patients have raised dissociative symptom levels and that their dissociative symptoms are related to objective EEG parameters. These findings are important because they may inspire sleep-related treatment methods for dissociative disorders.

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### 1. Introduction

Dissociative symptoms form a heterogeneous class of experiences varying from absent-mindedness, excessive daydreaming, and memory problems to confusion about one's own identity. In their most radical version, such symptoms define conditions like dissociative amnesia and depersonalization disorder. Given their stark heterogeneity, it is not surprising that dissociative disorders are among the most controversial nosological categories listed in the *Diagnostic and Statistical Manual of Mental Disorders*

(DSM-IV-TR) [1]. To date, there is no agreed-upon conceptualization of the taxonomy and aetiology of dissociative symptoms [2].

Epidemiological studies among psychiatric inpatients and outpatients have yielded prevalence estimates of severe dissociative symptoms, with rates usually exceeding 10% [3], while a recent epidemiological study in the UK general population found a prevalence rate of 0.95% [4]. Dissociative symptoms are not restricted to the dissociative disorders. Certain diagnostic groups, notably patients with borderline personality disorder, post-traumatic stress disorder (PTSD), obsessive compulsive disorder, and schizophrenia also display heightened levels of dissociative symptoms [5–7]. Prevalence rates of dissociative symptoms may also be raised in certain populations like, for example, homeless and runaway youths [8].

A recurrent theme in the clinical literature on dissociative symptoms is that they are caused by aversive experiences.

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More specifically, the idea is that dissociative symptoms like amnesia and derealisation help individuals to detach themselves from aversive life experiences [9]. Although there is correlational evidence to back up the link between dissociative symptoms and aversive life experiences [10], the question remains how such experiences might set into motion dissociative symptomatology.

An older idea that recently gained momentum focuses on sleep disturbances and how these disturbances might contribute to dissociative symptomatology. Thus, Levitan [11] hypothesized that “depersonalization is a compromise state between dreaming and waking” (p. 157). Recent studies have found a robust link between dissociation and self-reported sleep disturbances [12–14]. This finding has inspired some authors to speculate that a labile sleep–wake cycle may undermine cognitive efficiency and cause dreamlike mentation to emerge during waking consciousness, thereby fuelling dissociative symptoms [15]. There is some indirect evidence to support this hypothesis. For example, McNally and Clancy [16] relied on a sample of individuals who reported a history of child sexual abuse. In their sample, dissociative symptoms were more common in participants who had experienced sleep paralysis compared to those without such experiences. Dissociative symptoms also go along with increased frequencies of nightmare reports [17] and PTSD patients not only have raised levels of dissociative symptoms, but they also exhibit increased nightmare frequency and REM sleep density, and often suffer from insomnia [18].

Particularly interesting but under-researched is the potential link between REM sleep and dissociative symptoms. In her recent review, Llewellyn [19] stressed that REM sleep is usually accompanied by associative and visual hyperactivity so as to encode episodic memories. This author summarizes evidence to show that during REM sleep, the pre-frontal areas are in a state of deactivation resulting in fluid reasoning and bizarre thoughts. Thus, an excess of REM sleep and/or REM sleep activity throughout the day could lend dissociative qualities to cognitive functioning.

If sleep disturbances are a critical factor in the development of dissociative psychopathology, one would expect that patients with insomnia have heightened levels of dissociative symptoms. After all, the majority of patients who suffer from (primary) insomnia have a disturbed sleep–wake cycle [20]. In this study, we examined whether patients with insomnia have, indeed, raised scores on a validated questionnaire measuring dissociative symptoms. Additionally, and more exploratory, we investigated to what extent these symptoms are related to unusual sleep experiences, sleep quality, and EEG parameters. Previous studies relied on self-reported sleep disturbances. We were interested in whether the sleep–dissociation link is also evident when one looks at objective EEG indices, particularly REM sleep.

## 2. Methods and materials

### 2.1. Patients

The sample consisted of 45 consecutive inpatients (18 men, 28 women), who had been referred to Antwerp University Hospital, Belgium in the period between January 2010 and April 2010. Only patients with a diagnosis of primary insomnia were included. They had a variety of sleep complaints, such as having trouble with falling asleep, night-time awakenings, waking up early, and/or non-refreshing sleep. Mean age of the patients was 41.5 years ( $sd=13.68$ ; range: 17 to 78 years). Most of them were married or lived together with their partner (71.2%). A minority was either divorced (6.5%) or single (19.6%). Educational background ranged from limited (lower secondary education; 10.8%) to extended (higher education / university degree / postgraduate; 39.1%). Thirty-four patients (73.9%) were using medication during the study. Medication included benzodiazepines (21.7%), melatonin (2.2%), antidepressants (32.6%), pain medication (13%), and neuroleptics (4.3%). Twenty-eight percent of the patients suffered from a (self-reported) psychiatric disorder (e.g., mood-, or anxiety disorder) at the time of the study, and were unable to work due to their complaints for a mean of 22 days ( $sd=35.9$ ) over the last three months. On average, the current sample had not been able to execute their normal activities for 18.5 days ( $sd=34.0$ ) during the last three months. Eight patients (17.4%) had been hospitalized (mean duration: 1.98 days;  $sd=9.01$ ) in the last three months.

All patients had been referred for assessment of their persisting insomnia complaints. As part of the routine procedure, participants completed a number of self-report questionnaires (see Measures) during their stay of one or two nights in the specialized sleep clinic of Antwerp University Hospital. During their stay, data on sleep parameters were collected. For participants who underwent two night sessions ( $N=13$ , 28%), data of the second night were employed in analyses.

Patients provided written informed consent for the use of their data for the purpose of the present study. The study was approved by the standing ethical committee of the Antwerp University Hospital (B30020107809).

### 2.2. Measures

#### 2.2.1. Dissociative Experiences Scale (DES [21])

The DES is a self-report scale that requires participants to indicate on 100 mm visual analogue scales (anchors: 0=never; 100=always) to what extent they experience 28 dissociative experiences in daily life. Examples of such experiences include feelings of depersonalization, derealization, and psychogenic amnesia. Van IJzendoorn and Schuengel [22] provide meta-analytic evidence for the sound psychometric properties of the DES. In the current study, we calculated a mean total score of the 28 DES items.

### 2.2.2. Symptom Check List-90-Revised (SCL-90-R [23])

The SCL-90-R is a widely used self-report inventory measuring a broad range of psychopathological symptoms (e.g., depression, anxiety, somatic symptoms) in clinical as well as general population samples. In the current study, we used the validated Dutch version of the SCL-90-R [24] and calculated the Global Severity Index (GSI), which is the mean total score of the 90 SCL-90-R items.

### 2.2.3. Iowa Sleep Experiences Scale (ISES [12])

The ISES consists of 18 items asking the respondent to rate the frequency of various sleep-related and dream-related experiences on a 7-point Likert scale (anchors: 1 = never; 7 = several times a week). The ISES consists of two separate subscales: general sleep experiences (Cronbach's  $\alpha = .90$ , e.g., "I have recurring dreams") and lucid dreaming (Cronbach's  $\alpha = .83$ , e.g., "I am aware that I am dreaming, even as I dream"). The general sleep experiences subscale taps symptoms of narcolepsy, vivid and unusual dreams, and other distinct sleep experiences. The lucid dream subscale consists of several items that refer to the experience that one is aware of dreaming while still being asleep. Previous research by Watson [12] shows that these two subscales measure distinct constructs. Watson [12,25] obtained evidence for the convergent validity and internal consistency of the ISES.

### 2.2.4. Pittsburgh Sleep Quality Index (PSQI [26])

The PSQI is a self-report instrument measuring sleep quality and sleep aberrations during the last month. Consisting of 19 items, it measures seven aspects: subjective sleep quality, sleep latency, sleep efficiency, sleep time, sleep aberrations (e.g., nightmares, snoring, leg movements), medication use, and daytime problems (e.g., problems with staying awake during driving, eating, or social situations). The sum score of the 7 components yields a total score. Buysse and colleagues [26] examined the psychometric properties of the PSQI using an 18-month longitudinal design to monitor "good" and "bad" sleepers. They found an acceptable test–retest reliability and validity. The PSQI has reasonable sensitivity (89%) and specificity (85.5%) in differentiating between good and bad sleepers, making it a useful tool for clinical use as well as research [26].

### 2.2.5. Brussels Indices of Sleep Quality (BISQ [27])

The BISQ is a 29-item self-report instrument to index subjective feelings of sleep quality, and sleep parameters such as sleep efficiency, night-time awakenings, and sleepiness during the day. The instrument yields total scores for three subscales; 1) subjective sleep quality, 2) whether sleep quality is better or worse than usual, and 3) the subjective tolerance of sleep problems. Psychometric qualities of the measure are unknown as it has not been validated yet.

### 2.2.6. Traumatic Experiences Checklist (TEC [28])

The TEC is a reliable and valid self-report instrument to measure physical abuse, emotional abuse and neglect, sexual harassment, and sexual abuse, as well as other traumatic experiences. The TEC has especially been employed with psychiatric outpatients [28], but has also been administered to nonclinical samples in which low and skewed TEC scores are not unusual [29]. The correlation between the TEC and the Stressful Life Events Screening Questionnaire (SLESQ [30]) is relatively high,  $r = 0.77$ ,  $p < 0.01$ . In this study, we used the 26-item version of the TEC, which yields a total score between 0 and 26, with higher scores indicating higher levels of self-reported trauma.

## 2.3. Polysomnographic measures

Polysomnography during the night was performed, using electroencephalogram (EEG), electrooculography (EOG), and electromyogram (EMG) measurements (BrainRT, OSG, Rumst, Belgium). Electrodes for EEG registration were applied according to standard criteria of the American Academy of Sleep Medicine (AASM [31]), using frontal, central, and occipital head electrodes with reference electrodes at the mastoids. Central EEG (C4-A1; C3-A2), frontal EEG (C4-F4), and occipital EEG (A1-O2) recordings were obtained. EOG electrodes were placed at the outer canthi of the orbits and EMG was measured at the chin.

Sleep stages were scored according to the rules of the AASM [31]. This allowed determination of total sleep time (TST) defined as the total sleep period minus periods awake during this period, sleep efficiency index (SEI) defined as the ratio of TST/Time in Bed, sleep latency (SL), and relative duration of sleep stages all expressed as percentage of Sleep Period Time (SPT; time from sleep onset to last epoch of sleep). ECG was measured during the whole Time in Bed period. For the purpose of the current study, we focus on % sleep efficiency, sleep latency, % WASO (Wake After Sleep Onset), % REM SPT, % Stage 1 Sleep (S1), and ECG.

## 2.4. Statistical procedure

Statistical analyses were performed using SPSS 18.0 software. Cronbach's  $\alpha$  values were used to estimate internal consistency of the DES, SCL-90, ISES, PSQI, BISQ, and TEC. Pearson product–moment correlations were used to examine the links between the psychometric data and sleep parameters.

## 3. Results

Table 1 shows internal consistency and mean (sd) scores of all self-report measures as well as Pearson product–moment correlations between the measures.

Mean scores on DES were significantly higher compared to mean DES scores in an American/Canadian community sample ( $N = 415$ ;  $t = 4.13$ ,  $p < 0.001$  [33]). Mean scores on

Table 1

Mean scores and Pearson product–moment correlations between self-report measures ( $N=45$ ).

	<i>Cronbach's alpha</i>	<i>Mean (sd)</i>	<i>DES</i>	<i>SCL-90</i>	<i>ISES</i>	<i>PSQI</i>
<i>DES</i>	.94	14.23 (8.56)	–	–	–	–
<i>SCL-90</i>	.98	165.44 (46.43)	.51**	–	–	–
<i>ISES</i>	.90	44.98 (20.01)	.40**	.37*	–	–
<i>PSQI</i>	.63	9.50 (3.06)	.45**	.43**	.26	–
<i>TEC</i>	.82	2.67 (2.46)	.10	.39**	.49**	.39*

DES=Dissociative Experiences Scale; SCL-90=Symptom Checklist 90; ISES=Iowa Sleep Experiences Survey; PSQI=Pittsburgh Sleep Quality Index; TEC=Traumatic Experiences Checklist.

\*  $p<0.05$ .

\*\*  $p<0.01$ .

DES did not significantly differ from those obtained in a sample of 386 Dutch outpatients suffering from a variety of psychopathology ( $t=0.96$ ,  $p=0.34$  [32]). Four out of 46 participants (8.7%) displayed dissociation levels exceeding the clinical cut-off score for dissociative disorders (i.e., DES scores  $>30$  [34]).

SCL-90-R scores were comparable to those of primary care patients ( $M=164.3$ ,  $sd=49.9$ ,  $t=0.14$ ,  $p=0.89$  [35]), and significantly differed from normal controls ( $M=123.9$ ,  $sd=32.5$ ,  $t=7.16$ ,  $p<0.001$ ).

Significant correlations were found between self-reported dissociative symptoms (DES) and the SCL-90-R-GSI Scale, on the one hand, and the two sleep measures (ISES; PSQI), on the other hand, indicating that heightened dissociative levels and general psychopathology were related to poor sleep quality and unusual sleep experiences. Self-reported traumatic experiences (TEC) were not related to dissociation (DES), but were significantly related to SCL-90-R-GSI, unusual sleep experiences (ISES), and sleep quality (PSQI), suggesting that patients with a history of traumatic experiences showed elevated general psychopathology and unusual sleep experiences, as well as poor sleep quality.

Polysomnographic measures showed that patients had a mean sleep onset of 42 min ( $sd=38$ ; range: 10–210). Mean sleep onset latency was 23 min ( $sd=21$ ; range: 4.2–112.3), and mean number of minutes awake after sleep onset was 48 min ( $sd=37$ ; range: 1–164). Their subjective estimate of the hours spent asleep was 5.2 h ( $sd=1.9$  h; range: 2–10 h).

Table 2 displays the Pearson product–moment correlations between self-report measures and objective sleep parameters obtained during polysomnographic recordings. As expected, the DES correlated positively with % REM SPT. Because antidepressants may affect REM sleep, we conducted an independent samples  $t$ -test to compare the % REM SPT in patients using medication, and patients not on medication. Less time spent in REM sleep was not related to the use of antidepressant medication ( $t(42)=-1.07$ ,  $p=0.29$ ). Also, the DES correlated negatively with time spent awake during sleep period time. Finally, ISES correlated significantly and positively with mean ECG rate and with total REM time in minutes.

Table 2

Pearson product–moment correlations between self-report measures and sleep parameters ( $N=45$ ).

	<i>DES</i>	<i>SCL-90</i>	<i>ISES</i>	<i>PSQI</i>	<i>TEC</i>
% sleep efficiency	.27	.14	.21	–.12	.24
Sleep onset	–.05	.02	.04	–.10	–.12
WASO	–.32*	–.21	–.26	.18	–.26
Total REM	.26	.22	.34*	.01	.20
% REM SPT	.31*	.19	.23	.05	.06
% S1 SPT	–.12	–.04	.11	.07	.26
ECG	.22	.26	.41**	.10	.29

DES=Dissociative Experiences Scale; SCL-90=Symptom Checklist 90; ISES=Iowa Sleep Experiences Survey; PSQI=Pittsburgh Sleep Quality Index; TEC=Traumatic Experiences Checklist; Sleep onset=sleep onset latency in minutes; WASO=wake after sleep onset, percentage awake during the whole sleep period (SPT=sleep period time); Total REM=total REM sleep in minutes; % REM SPT=percentage REM sleep during the whole sleep period; % S1 SPT=percentage Stage 1 sleep during sleep period time; ECG=mean score on electrocardiogram.

\*  $p<0.05$ .

\*\*  $p<0.01$ .

Participants rated the sleep quality items of the BISQ on scales ranging from 1 (very poor) to 10 (excellent). Their mean rating was 5.2 ( $sd=1.82$ ). As shown in Table 3, the BISQ correlated with several objective sleep parameters, indicating that patients' subjective experience of sleep quality was in line with objective measures such as sleep efficiency, total sleep time (in minutes), time spent awake, time in REM, and time in S1 [27].

Patients were also asked to indicate on a 5-point Likert scale (range: 0='not at all sleepy', to 4='very sleepy') how sleepy they felt upon awakening. Many of them (61 %) scored '2' or more, indicating that they felt 'rather sleepy' to 'very sleepy'. A majority (73%) of the patients said that

Table 3

Pearson product–moment correlations between subjective sleep measure (BISQ; horizontal row) and objective sleep parameters (polysomnography; left column;  $N=45$ ).

	Sub. sleep onset	Time wake during night	Subjective TST	Sleep quality (1–10)
<i>Sleep onset</i>	.34*	.18	–.01	–.25
<i>SOL</i>	.41**	.01	.03	–.05
<i>SPT</i>	–.33*	–.04	.38*	–.04
<i>TST</i>	–.51**	–.36*	.52**	.25
<i>SE %</i>	–.57**	–.50**	.42**	.42**
<i>SPT wake</i>	.31*	.65**	–.37*	–.38*
<i>REM TST</i>	–.24	.47**	.33*	.09
<i>S1 TST</i>	–.10	–.12	.45**	.38*
<i>S2 TST</i>	–.44**	–.25	.45**	.17
<i>ECG</i>	–.04	–.10	.18	–.01

SOL=Sleep onset latency; SPT=Sleep Period Time; TST=Total Sleep Time; SE %=Sleep Efficiency in Percentage; SPT Wake=minutes awake during sleep period time; REM TST=minutes of REM sleep during total sleep time; S1 TST=minutes of Stage 1 sleep during total sleep time; S2 TST=minutes of Stage 2 sleep during total sleep time; ECG=mean score on electrocardiogram.

\*  $p<0.05$ .

\*\*  $p<0.01$ .



their sleep complaints were unacceptable to bear. Sleepiness upon awakening was significantly correlated with DES scores,  $r=0.45$ ,  $p<0.01$ . As shown in Table 3, objective sleep efficiency correlated positively with subjective total sleep time, and subjective sleep quality. Moreover, objective time spent awake during the night correlated negatively with subjective time spent awake and subjective sleep quality.

#### 4. Discussion

This study is – to the best of our knowledge – the first to examine dissociative symptoms in patients with insomnia. Before we discuss the conclusions that can be drawn from our findings, it is important to emphasize some limitations of the current study. Although our sample consisted of a unique and rather homogeneous group of patients with insomnia, its size was relatively small. Thus, future research might want to include a larger and more heterogeneous sample of patients with sleep problems, preferably also outpatients. Furthermore, medication did not seem to have affected our results, as there was no statistically significant difference between patients who used medication and those who did not. Nevertheless, follow-up research should address the issue of medication and how it relates to the dissociation–sleep link in a more systematic way. Most importantly, our study relied on a cross-sectional design that precludes any causal interpretations. Also, all measures were moderately related to each other, with the exception of ISES and PSQI. This pattern makes it difficult to formulate specific hypotheses. Thus, a longitudinal set-up in which patients undergoing targeted treatments for their sleep dysfunctions are followed over time would provide a more optimal starting point for testing causal hypotheses.

With these limitations in mind, the main findings of our study can be catalogued as follows. First, we found that as a group, patients suffering from insomnia had heightened dissociative levels, which is in keeping with research showing a solid link between sleep experiences and dissociative symptoms in non-clinical samples [12,36–38].

Second, dissociative symptoms did not correlate with self-reports of traumatic experiences, but trauma was positively related to the two sleep measures (ISES and PSQI). This is in accordance with the idea that sleep disturbances rather than traumatic experiences per se act as stage setter for dissociative symptoms. Because our study was cross-sectional in nature, it does not allow for strong causal conclusions. However, one distinct possibility is that specifically aversive childhood experiences rather than aversive experiences in general, might lead to sleep disturbances, which in turn might serve as the more proximal antecedents of dissociative symptoms. This interpretation might reconcile seemingly conflicting theories about the origins of dissociative symptoms, with some theories emphasizing the traumatic origins of dissociative symptoms

and other theories stressing the etiological role of sleep disturbances [2,9,39].

Third, we found that subjective sleep evaluations of insomnia patients often correlate with objective sleep parameters, which supports the integrity of the subjective sleep data obtained in this group.

Fourth and most importantly, our study is the first to document that raised dissociative scores are related not only to subjective sleep parameters, but also to objective sleep indices. More specifically, we found that dissociative symptoms are associated with increased periods of sleep time spent in REM sleep, and with more sleepiness at awakening. This is reminiscent of recent literature about REM sleep and psychopathology. For example, Levin and Nielsen [40] emphasized the concept of “cross-state continuity,” which assumes that “...some structures and processes implicated in nightmare production are also engaged during the expression of pathological signs and symptoms during the waking state” (p. 483). Thus, an influx of dreamlike mentation during the day may fuel dissociative symptoms. A related view is the notion of transliminality [41], which assumes that there exist robust individual differences in the extent to which mentation may cross thresholds into and out of consciousness. Using a self-report scale that intends to measure this trait – the Revised Transliminality Scale (RTS) – Soffer-Dudek and Shahar [42] showed in a longitudinal study that people who score high on transliminality (i.e., who are attuned to their inner fantasy life) subsequently report more unusual sleep experiences than those who score low on this trait.

There is consensus that REM sleep is important for emotional memory formation [43]. For instance, Crick and Mitchison [44] emphasized that REM sleep plays a role in memory consolidation. Our finding that dissociative symptoms are related to more intense REM periods might reflect heightened cognitive effort to cope with irrelevant and noisy memory traces, especially when the memory is emotional in nature [43]. Also germane to this issue is the conceptualization of Llewellyn [19]. This author summarizes evidence to show that REM dreaming is more perceptually vivid and hyperassociational compared to the waking state, and argues that during dreaming there is an enhanced access to remote memory material. REM dreaming may therefore provide the ideal state for elaborative encoding of emotional memories. However, when dreaming and waking states become de-differentiated, it is conceivable that the hyperassociational mentation may intrude consciousness and contribute to dissociative experiences. Note that we observed in previous studies [14,45] a connection between dissociative symptoms and narcoleptic experiences, which represent a paradigmatic example of de-differentiation of dreaming and waking. Clearly, disturbed sleep–wake cycles bear relevance to clinical practice, as is also demonstrated by case reports about patients with narcolepsy who misinterpret their dreamlike hallucinatory experiences as real events and

sincerely believe that they have been the victim of sexual assault or another offence [45].

Fifth, dissociation was related to less time spent awake during the night. This contradicts an earlier study that found dissociative symptoms in a healthy sample to be related to more fragmented sleep [13]. We have no ready explanation for these conflicting findings but they might be explained by the different samples (patients with insomnia versus healthy individuals), the different methods for measuring fragmented sleep in the two studies, and the different context (psychological laboratory versus sleep laboratory). Also, it is well known among clinicians that patients suffering from insomnia tend to sleep better during recordings in the hospital than they sleep at home.

Finally, a higher heart rate during the night correlated with a higher score on unusual sleep experiences (ISES). This is intuitively plausible because it stands to reason that patients who experience more sleep aberrations during the night react with elevated arousal responses to such episodes, although the causal relation may of course be the other way around. In general, then, it appears that dissociative symptoms are more related to the cognitive aspects of sleep (i.e., REM sleep), while self-reported sleep disturbances are primarily related to the arousal aspects of sleep.

In conclusion, our finding that dissociation levels are elevated in patients suffering from insomnia and are related to various sleep parameters suggests several avenues for future research. Research that addresses the sleep–dissociation link in clinical samples is urgently needed, because most previous studies have relied on student samples. Future studies might elucidate the type of sleep architecture that is most reliably associated with different dissociative disorders in a longitudinal design, and then establish remediation programs, including medication regimens, to address underlying sleep deficits and irregularities. This would be an entirely novel and exciting lead.

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